STIC Search

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FILE 'HCAPLUS' ENTERED AT 18:36:31 ON 08 JUN 2003

L1 1314 SEA ABB=ON PDD OR ((?PERVASIVE? OR ?CHILD?)(3A)?DEVELOP? OR ?DYSAUTONOMIC?)(W)?DISORDER? OR ?AUTISM? OR ?AUTISTIC?(W)?DISORDER?

L2 2 SEA ABB=ON L1 AND (?STOOL? OR ?FECES?(W)?IMMUNOASSAY?)

L3 10 SEA ABB=ON L1 AND (?BIOMARKER? OR (?BIOLOGICAL? OR ?COMPOUND?)(2A)?MARKER)

L4 10 SEA ABB=ON L2 OR L3

L5 1 SEA ABB=ON L4 AND (H OR ?HELICOBACTER?)(W)?PYLORI?

D AU

L6 10 SEA ABB=ON L4 OR L5 10 cits from CAPlus

FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT 18:43:24 ON 08 JUN 2003

L7 38 SEA ABB=ON L6

L8 31 DUP REMOV L7 (7 DUPLICATES REMOVED) 31 cits from "other dbs"

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=> d que stat 16
L1          1314 SEA FILE=HCAPLUS ABB=ON  PDD OR ((?PERVASIVE? OR ?CHILD?)(3A)?D
                 EVELOP? OR ?DYSAUTONOMIC?)(W)?DISORDER? OR ?AUTISM? OR
                 ?AUTISTIC?(W)?DISORDER?
L2             2 SEA FILE=HCAPLUS ABB=ON  L1 AND (?STOOL? OR ?FECES?(W)?IMMUNOAS
                 SAY?)
L3            10 SEA FILE=HCAPLUS ABB=ON  L1 AND (?BIOMARKER? OR (?BIOLOGICAL?
                 OR ?COMPOUND?)(2A)?MARKER)
L4            10 SEA FILE=HCAPLUS ABB=ON  L2 OR L3
L5             1 SEA FILE=HCAPLUS ABB=ON  L4 AND (H OR ?HELICOBACTER?)(W)?PYLORI
                 ?
L6            10 SEA FILE=HCAPLUS ABB=ON  L4 OR L5


=> d ibib abs 16 1-10

L6   ANSWER 1 OF 10  HCAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2003:300431  HCAPLUS
DOCUMENT NUMBER:          138:319703
TITLE:                    Chemically modified erythropoietin lacking some
                          effects on bone marrow for protection, restoration,
                          and enhancement of responsive cells, tissues and
                          organs
INVENTOR(S):              Brines, Michael; Cerami, Antony; Cerami, Carla
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 63 pp., Cont.-in-part of Appl.
                          No. PCT/US01/49479.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ----------------  ----  --------      ---------------  --------
     US 2003072737      A1   20030417      US 2002-188905   20020703
     US 2002086816      A1   20020704      US 2000-753132   20001229
     US 6531121         B2   20030311
     WO 2002053580      A2   20020711      WO 2001-US49479  20011228
         W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
            CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
            GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
            LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
            PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
            UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
            CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
            BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:                  US 2000-259245P  P  20001229
                                        US 2000-753132   A2 20001229
                                        WO 2001-US49479  A2 20011228

AB   Methods and compns. are provided for protecting or enhancing a responsive
     cell, tissue, organ or body part function or viability in vivo, in situ or
     ex vivo in mammals, including human beings, by systemic or local
     administration of a tissue protective cytokine.  The tissue protective
     cytokine is a chem. modified erythropoietin, e.g. acetylated,
     biotinylated, carbamylated, succinylated, desialylated, guanidinated,
     amidated, trinitrophenylated, or nitrated EPO.  The tissue protective
     cytokine lacks activity selected from the group consisting of increasing
     hematocrit, vasoconstriction, hyperactivating platelets, procoagulant
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activities and increasing prodn. of thrombocytes. The tissue protective cytokine is capable of traversing an endothelial cell barrier, e.g. the blood-brain barrier, the blood-eye barrier, the blood-testes barrier, the blood-ovary barrier, and the blood-uterus barrier. The tissue protective cytokine is therefore useful for protecting cell from injury caused by seizure, multiple sclerosis, stroke, hypotension, cardiac arrest, ischemia, myocardial infarction, inflammation, age-related loss of cognitive function, radiation damage, cerebral palsy, neurodegenerative disease, Alzheimer's disease, Parkinson's disease, Leigh disease, AIDS dementia, memory loss, amyotrophic lateral sclerosis, alcoholism, mood disorder, anxiety, attention deficit, **autism**, Creutzfeld-Jakob disease, brain or spinal cord trauma or ischemia, heart-lung bypass, chronic heart failure, macular degeneration, diabetic neuropathy, diabetic retinopathy, glaucoma, retinal ischemia or retinal trauma.

L6 ANSWER 2 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:739852 HCAPLUS
DOCUMENT NUMBER: 138:37295
TITLE: Elevated plasma gamma-aminobutyric acid (GABA) levels in austistic youngsters: stimulus for a GABA hypothesis of **autism**
AUTHOR(S): Dhossche, Dirk; Applegate, Heather; Abraham, Ann; Maertens, Paul; Bland, Lorna; Bencsath, Aladar; Martinez, Jose
CORPORATE SOURCE: Department of Psychiatry, University of South Alabama, AL, USA
SOURCE: Medical Science Monitor (2002), 8(8), PR1-PR6
CODEN: MSMOFR; ISSN: 1234-1010
PUBLISHER: Medical Science International Publishing Co., Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Autistic Disorder** is an early-onset developmental disorder with severe lifelong impact on social functioning, communication, and behavior. There is currently no marker or cure. The pathophysiol. and etiol. are obscure. Evidence for abnormal GABA function in **Autistic Disorders** is limited. A few case-reports and small studies have reported differences in GABA levels in plasma, platelets, and urine, compared to controls. Further studies on abnormalities of GABA function in **Autistic Disorder** are warranted. Plasma GABA levels were measured using a new and sensitive technique, based on gas chromatog./mass spectrometry, in a small group of youngsters with **Autistic Disorder** and Attention-Deficit/Hyperactivity Disorder. Participants were outpatients between ages 5-15, satisfying modern criteria for these disorders. Elevated plasma GABA levels were found in youngsters with **Autistic Disorder**. Psychotropic medications did not seem to affect plasma GABA levels in this study. Plasma GABA levels decreased with age. Conclusions: Elevated plasma GABA levels may be a biochem. marker of **Autistic Disorder**. This study supports the hypothesis that GABAergic mechanisms play a role in the etiol. or pathophysiol. of **Autistic Disorder**. However, the hypothesis remains unspecified owing to lack of research. Future studies on the clin. assocns. of seizure disorders, mood disorders, and catatonia in autistic people may provide the necessary data to formulate a coherent theory of GABA dysfunction in **Autistic Disorder**. More trials of medication with known or suspected effects on GABA function are warranted.
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 10 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:696162 HCAPLUS
DOCUMENT NUMBER: 137:210935
TITLE: Methods, systems and computer program products for determining the biological effect and/or activity of drugs, chemical substances and/or pharmaceutical compositions based on their effect on the methylation status of the DNA
INVENTOR(S): Olek, Alexander; Berlin, Kurt
PATENT ASSIGNEE(S): Epigenomics AG, Germany
SOURCE: PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002070741	A2	20020912	WO 2002-EP2254	20020301

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-272484P P 20010301
AB This invention is related to methods, systems and computer program products for detg. the biol. effect and/or activity of drugs, chem. substances and/or pharmaceutical compns. using their effect on DNA methylation as a **marker** for their **biol.** effect(s). The invention is further related to the use of the inventive methods, systems and computer program products in obtaining new biol. active compds. which can be used as so-called "lead" compds. for new and effective medicaments and treatment strategies of, in particular, human diseases.

L6 ANSWER 4 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:488136 HCAPLUS
DOCUMENT NUMBER: 137:30245
TITLE: Methods for diagnosing **pervasive development disorders**, dysautonomia and other neurological conditions
INVENTOR(S): Fallon, Joan M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002081628	A1	20020627	US 2001-990909	20011116

PRIORITY APPLN. INFO.: US 2000-249239P P 20001116
AB Methods for aiding in the diagnosis of disorders including, but not limited to, PDDs (**Pervasive Development**

Disorders), **Dysautonomic disorders**, Parkinson's disease and SIDS (Sudden Infant Death Syndrome). In one aspect, a diagnosis method comprises analyzing a **stool** sample of an individual for the presence of a **biol. marker** (or **marker compd.**) comprising one or more pathogens, which provides an indication of whether the individual has, or can develop, a disorder including, but not limited to, a **PDD**, Dysautonomia, Parkinsons disease and SIDS. Preferably, the presence of one or more pathogens is detd. using a **stool** immunoassay to det. the presence of antigens in a **stool** sample, wherein such antigens are assocd. with one or more pathogens including, but not limited to, Giardia, Cryptosporidium, E. histolytica, C. difficile, Adenovirus, Rotavirus or **H. pylori**.

L6 ANSWER 5 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:142908 HCAPLUS
DOCUMENT NUMBER: 136:180178
TITLE: Methods for diagnosing and treating dysautonomia and other dysautonomic conditions
INVENTOR(S): Fallon, Joan M.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002014537	A2	20020221	WO 2001-US25343	20010814
WO 2002014537	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001084865	A5	20020225	AU 2001-84865	20010814
US 2002037284	A1	20020328	US 2001-929592	20010814
PRIORITY APPLN. INFO.:			US 2000-224991P P	20000814
			WO 2001-US25343 W	20010814

AB Methods for aiding in the diagnosis of **dysautonomic disorders** and dysautonomic conditions and methods for treating individuals diagnosed as having a **dysautonomic disorder** or a dysautonomic condition. In one aspect, a diagnosis method comprising analyzing a **stool** sample of an individual for the presence of a **biol. marker** wherein the quantity of the **biol. marker** is an indication of whether the individual has, or can develop, a dysuatonic disorder or dysautonomic condition, as well as a therapeutic method for treating a **dysautonomic disorder** or dysautonomic condition by administration of, e.g., secretin, neuropeptides, peptides and/or digestive enzymes.

L6 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:696725 HCAPLUS
DOCUMENT NUMBER: 136:245657

TITLE: Toward a biology of **autism**: possible role of certain neuropeptides and neurotrophins
AUTHOR(S): Nelson, Karin B.
CORPORATE SOURCE: Neuroepidemiology Branch, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, 20892-1447, USA
SOURCE: Clinical Neuroscience Research (2001), 1(4), 300-306
CODEN: CNRLBU; ISSN: 1566-2772
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Autism** is a behaviorally defined syndrome for which there is no known **biol. marker.** Although **autism** is thought to be a disorder of brain development, there were few efforts to study early regulators of brain development in this disorder. This paper describes a recent study of neonatal blood of children with later-diagnosed autistic spectrum disorders, comparing them with 2 groups of affected children, those with mental retardation without **autism** , or with cerebral palsy, and unaffected control children, using recycling immunoaffinity chromatog. We measured concns. of 4 neuropeptides and 4 neurotrophins, finding that neonatal concns. of the neuropeptides vasoactive intestinal peptide, calcitonin gene-related peptide, and the neurotrophins brain derived neurotrophic factor and neurotrophin 4/5 were higher in children in the autistic spectrum, and in those with mental retardation without **autism**, than in children with cerebral palsy or healthy control children. In 99% of children with **autism** and 97% with mental retardation, levels of at least one of these substances exceeded those of all control children. Concns. were similar in subgroups of the autistic spectrum (core syndrome with or without mental retardation, other autistic spectrum disorders with or without mental retardation), and in the presence or absence of a history of regression. 2 Other neuropeptides and 2 neurotrophins were present in similar concns. in all groups examd. Thus overexpression of certain neuropeptides and neurotrophins was obsd. in neonatal blood of children with later diagnoses of **autism** or cognitive disability.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:666361 HCAPLUS
DOCUMENT NUMBER: 132:220666
TITLE: Normal cerebrospinal fluid glutathione concentrations in Parkinson's disease, Alzheimer's disease and multiple system atrophy
AUTHOR(S): Konings, C. H.; Kuiper, M. A.; Teerlink, T.; Mulder, C.; Scheltens, P.; Wolters, E. C.
CORPORATE SOURCE: Department of Clinical Chemistry, Academisch Ziekenhuis Vrije Universiteit, Amsterdam, 1007 MG, Neth.
SOURCE: Journal of the Neurological Sciences (1999), 168(2), 112-115
CODEN: JNSCAG; ISSN: 0022-510X
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We measured total glutathione concns. in the cerebrospinal fluid (CSF) of non-demented Parkinson's disease patients (PD; n=71), demented PD patients (**PDD**; n=13), multiple system atrophy patients (MSA; n=10), Alzheimer's disease patients (AD; n=17) and age-matched controls (n=21). No statistically significant differences in the mean total CSF glutathione

concns. were found between groups and dopaminomimetic treatment was not found to have any effect on total CSF glutathione levels. Our main conclusion is that total glutathione is not useful as a CSF marker for assumed oxidative stress in patients with PD, MSA or AD.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:103982 HCAPLUS
DOCUMENT NUMBER: 130:295008
TITLE: Whole blood serotonin and plasma beta-endorphin in autistic probands and their first-degree relatives
AUTHOR(S): Leboyer, Marion; Philippe, Anne; Bouvard, Manuel; Guilloud-Bataille, Michel; Bondoux, Dominique; Tabuteau, Francois; Feingold, Josue; Mouren-Simeoni, Marie-Christine; Launay, Jean-Marie
CORPORATE SOURCE: INSERM U155, Faculte Jussieu, Paris, Fr.
SOURCE: Biological Psychiatry (1999), 45(2), 158-163
CODEN: BIPCBF; ISSN: 0006-3223
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Whole blood serotonin (5-HT) and C-terminally directed .beta.-endorphin protein immunoreactivity (C-ter-.beta.-EP-ir) are known to be elevated in autistic subjects and might be possible markers of genetic liability to **autism**. This study thus investigates the familial aggregation of 5-HT and of C-ter-.beta.-EP-ir levels in first degree relatives of autistic probands. In a sample of 62 autistic subjects and 122 of their first-degree relatives, compared to age and sex-matched controls, we measured 5-HT by radioenzymol. and C-ter-.beta.-EP-ir by RIA. We confirm the previously reported familiarity of hyperserotoninemia in **autism** as mothers (51%), fathers (45%) and siblings (87%) have elevated levels of 5-HT, and we reveal presence of elevated levels of C-ter-.beta.-EP-ir in mothers (53%) of autistic subjects. Familial aggregation of quant. variables, such as concn. of neurotransmitters, within unaffected relative could serve as an intermediate phenotype and might thus help the search of genetic susceptibility factors in **autism**.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:150861 HCAPLUS
DOCUMENT NUMBER: 128:255939
TITLE: Platelet serotonin levels as a **biological marker** for **autism**
AUTHOR(S): Quinhones Levy, Pilar; Pires Bicho, Manuel
CORPORATE SOURCE: Laboratorio de Genetica, Faculdade de Medicina de Lisboa, Universidade de Lisboa, Lisbon, Port.
SOURCE: Acta Medica Portuguesa (1997), 10(12), 927-931
CODEN: AMPOD2; ISSN: 0253-0562
PUBLISHER: Ordem dos Medicos
DOCUMENT TYPE: Journal
LANGUAGE: Portuguese

AB Platelet levels of serotonin were detd. by a quant. direct RIA, in a group of autistic patients and a control group. Thirty six autistic patients (28 males and 8 females), all with severe mental retardation, and a group of 23 matched controls, were studied. The serotonin levels in autistic patients (88.37 mmol/dL) were higher than in the control group (49.54 mmol/dL). There were no differences in levels between the sexes and age

groups among subjects in the patient and the control groups. The authors detected a hyperserotoninemia in 70% of the autistic patients. The authors also discuss correlations between serotonin levels in the patients with known etiologies and levels quoted in the literature and propose RIA to be used as a quick, easy, and reliable method for the anal. of large nos. of samples.

L6 ANSWER 10 OF 10 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:735890 HCAPLUS
DOCUMENT NUMBER: 127:328695
TITLE: Diagnosis of **autism** and treatment therefor
INVENTOR(S): Shaw, William
PATENT ASSIGNEE(S): Children's Mercy Hospital, USA
SOURCE: U.S., 38 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5686311	A	19971111	US 1995-494200	19950623

PRIORITY APPLN. INFO.: US 1995-494200 19950623

AB A method for diagnosing the likelihood of **autism** in patients is provided which comprises first obtaining from the patient a sample of body fluid such as urine and analyzing the sample to det. the quantity therein of at least one **marker compd.** selected from the group consisting of citramalic acid, 5-hydroxy-methyl-2-furoic acid, 3-oxo-glutaric acid, furan-2,5-dicarboxylic acid, tartaric acid, arabinose, dihydroxyphenylpropionic acid, and phenylcarboxylic acid; if the quantities of one or more of the compds. are abnormally high, as compared with the urine of non-autistic individuals, an ultimate diagnosis of **autism** is likely. The invention also pertains to a method of treating autistic patients by administration of antifungal drugs, to ameliorate the clin. symptoms of **autism.**

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=> d que stat l8
L1          1314 SEA FILE=HCAPLUS ABB=ON  PDD OR ((?PERVASIVE? OR ?CHILD?)(3A)?D
                 EVELOP? OR ?DYSAUTONOMIC?)(W)?DISORDER? OR ?AUTISM? OR
                 ?AUTISTIC?(W)?DISORDER?
L2             2 SEA FILE=HCAPLUS ABB=ON  L1 AND (?STOOL? OR ?FECES?(W)?IMMUNOAS
                 SAY?)
L3            10 SEA FILE=HCAPLUS ABB=ON  L1 AND (?BIOMARKER? OR (?BIOLOGICAL?
                 OR ?COMPOUND?)(2A)?MARKER)
L4            10 SEA FILE=HCAPLUS ABB=ON  L2 OR L3
L5             1 SEA FILE=HCAPLUS ABB=ON  L4 AND (H OR ?HELICOBACTER?)(W)?PYLORI
                 ?
L6            10 SEA FILE=HCAPLUS ABB=ON  L4 OR L5
L7            38 SEA L6
L8            31 DUP REMOV L7 (7 DUPLICATES REMOVED)

=> d l8 ibib abs 1-31

L8   ANSWER 1 OF 31  EMBASE  COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER:   2003136894  EMBASE
TITLE:              Childhood autism: A circuit syndrome?.
AUTHOR:             Lee D.A.; Lopez-Alberola R.; Bhattacharjee M.
CORPORATE SOURCE:   Dr. D.A. Lee, Department of Psychiatry, Tulane University
                    School of Medicine, 1430 Tulane Avenue, New Orleans, LA
                    70112, United States. dlee@tulane.edu
SOURCE:             Neurologist, (2003) 9/2 (99-109).
                    Refs: 174
                    ISSN: 1074-7931 CODEN: NROLFW
COUNTRY:            United States
DOCUMENT TYPE:      Journal; General Review
FILE SEGMENT:       007     Pediatrics and Pediatric Surgery
                    008     Neurology and Neurosurgery
                    032     Psychiatry
                    037     Drug Literature Index
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   BACKGROUND - Autism is a disorder that can lead to life-long
     disability. Currently, the etiology of autism is unknown, and
     although there are treatments for some of the behavioral abnormalities,
     there is no cure. REVIEW SUMMARY - While this article will review the
     clinical, anatomic, and pathologic features seen in autism, the
     primary focus will be to present a new and provocative unifying theory
     regarding the underlying mechanisms causing this disorder. Current
     research advances, some controversial, will be discussed, and a novel
     definition of autism as a "circuit syndrome" will be presented.
     The work elaborated here will tie many of the disparate findings together,
     based on the idea that autism arises from abnormalities of the
     cerebellolimbic circuitry. Some of the more alternative theories of
     autism, such as mercury toxicity, linkage to the measles, mumps,
     and rubella vaccine, and the use of secretin will be discussed. Finally,
     pharmacologic treatment options will be reviewed. CONCLUSIONS -
     Autism is not single disorder but represents dysfunction of the
     cerebellolimbic circuitry that can arise from many different etiologies.

L8   ANSWER 2 OF 31  EMBASE  COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER:   2003091195  EMBASE
TITLE:              RG-1068 RepliGen.
AUTHOR:             Wheeler G.
CORPORATE SOURCE:   G. Wheeler, 102-711 West 17th Ave., Vancouver, BC V5Z 1V1,
                    Canada. editor@greyowltutor.com
SOURCE:             Current Opinion in Investigational Drugs, (1 Jan 2003) 4/1
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(66-71).
Refs: 26
ISSN: 1472-4472 CODEN: CIDREE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
038 Adverse Reactions Titles
007 Pediatrics and Pediatric Surgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB RG-1068 is a synthetic form of the natural human hormone secretin under development by RepliGen for the potential treatment of **autism**. RG-1068 received Fast Track designation for the treatment of pediatric **autism** in September 2001, and in February 2002, it entered phase III clinical trials.

L8 ANSWER 3 OF 31 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-723268 [78] WPIDS
DOC. NO. CPI: C2002-204757
TITLE: Determination of the biological effect and/or activity of at least one drug, chemical substance and/or pharmaceutical composition involves using their effect on DNA-methylation as a **marker** for their **biological** effects.
DERWENT CLASS: B04 D16
INVENTOR(S): BERLIN, K; OLEK, A
PATENT ASSIGNEE(S): (EPIG-N) EPIGENOMICS AG
COUNTRY COUNT: 99
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002070741	A2	20020912	(200278)*	EN	86

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002070741	A2	WO 2002-EP2254	20020301

PRIORITY APPLN. INFO: US 2001-272484P 20010301
AN 2002-723268 [78] WPIDS
AB WO 200270741 A UPAB: 20021204
NOVELTY - Determining biological effect and/or activity of a drug, is new.
DETAILED DESCRIPTION - Determining the biological effect and/or activity of at least one drug, chemical substance and/or pharmaceutical composition involving:
(a) obtaining a biological sample A containing DNA from at least one individual, tissue, cell or other biological material containing DNA, which was exposed to the at least one drug, chemical substance and/or

pharmaceutical composition;

(b) obtaining a biological sample B containing DNA from at least one individual, tissue, cell or other biological material containing DNA, which was not exposed to the at least one drug, chemical substance or pharmaceutical composition;

(c) analyzing the level of cytosine methylation at chosen sites of the DNA contained in the samples A and B;

(d) selecting the sites which are differentially methylated between the DNA in samples A and B to generate a knowledge base; and

(e) concluding from the knowledge base on the biological effect and/or activity of the at least one drug, chemical substance or pharmaceutical composition.

ACTIVITY - Cytostatic; Antipsychotic; Nootropic; Cardiant; Gastrointestinal-Gen.; Antiinflammatory; Antibacterial; Vulnerary; Dermatological; Osteopathic; Hypotensive; Antiulcer; Anticonvulsant.

MECHANISM OF ACTION - None given.

USE - For determining the biological effect and/or activity of at least one drug, chemical substance and/or pharmaceutical composition in a biological sample, such as a eukaryotic and/or prokaryotic cell line, a biopsy sample, blood, sputum, faeces, urine, cerebral liquid, tissue embedded in paraffin, tissue derived from eyes, intestine, brain, heart, prostata, kidney, lung, breast or liver and/or histological samples. The biologically effective and/or active drug, chemical substance and/or pharmaceutical composition is useful for the treatment of a disease and/or medical condition such as unwanted side effects of medicaments, cancers, dysfunctions, damages or diseases of the central nerval system (CNS), aggressive symptoms or behavioral disorders, clinical, psychological and social consequences of brain injuries, psychotic disorders and disorders of the personality, dementia and/or associates syndromes, cardiovascular diseases, malfunctions or damages, diseases, malfunctions or damages of the gastrointestine, diseases, malfunctions or damages of the respiratory system, injury, inflammation, infection, immunity and reconvalescence, diseases, malfunctions or damages as consequences of modifications in the developmental process, diseases, malfunctions or damages of the skin, muscles, connective tissue or bones, endocrine or metabolic diseases, malfunctions or damages, headache and sexual malfunctions; especially located in methylation relevant regions of genes related with leukemia, head and eck cancer, Hodgkin's disease, gastric cancer, prostate cancer, renal cancer, bladder cancer, breast cancer, Burkitt's lymphoma, Wilm's tumor, Prader- Willi/ Angelman syndrome, ICF syndrome, dermatofibroma, hypertension, pediatric neurobiological diseases, **autism**, ulcerative colitis, fragile X syndrome, and Huntington's disease) (all claimed).

ADVANTAGE - The method provides a necessary second step in the combinatorial chemistry screening, after the potential target/lead compound has been identified/found, to further confirm/determine the biological effect of the compound in an in vivo context, by providing a method to generate and evaluate epigenotypic information on a large scale using pattern of methylation sensitive sites in genes.

Dwg.0/0

L8 ANSWER 4 OF 31 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2002-257612 [30] WPIDS

DOC. NO. CPI: C2002-076714

TITLE: Determining if an individual has, or can develop, **dysautonomic disorder** by analyzing pancreatic or digestive enzyme in **stool** sample of individual, and correlating analysis of enzyme with a **dysautonomic disorder.**

DERWENT CLASS: B04 D16

INVENTOR(S): FALLON, J M
PATENT ASSIGNEE(S): (FALL-I) FALLON J M
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002014537	A2	20020221	(200230)*	EN	26

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

US 2002037284 A1 20020328 (200230)
AU 2001084865 A 20020225 (200245)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002014537	A2	WO 2001-US25343	20010814
US 2002037284	A1 Provisional	US 2000-224991P	20000814
		US 2001-929592	20010814
AU 2001084865	A	AU 2001-84865	20010814

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001084865	A Based on	WO 200214537

PRIORITY APPLN. INFO: US 2000-224991P 20000814; US 2001-929592 20010814

AN 2002-257612 [30] WPIDS

AB WO 200214537 A UPAB: 20020513

NOVELTY - Determining (M1) if an individual has, or can develop, a **dysautonomic disorder** (DD) or condition, by obtaining a **stool** sample from the individual, analyzing a compound (e.g. pancreatic or digestive enzyme) in the **stool** sample, and correlating the analysis of the compound with a **dysautonomic disorder** or condition or the absence of the disorder.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for treating (M2) DD by administering secretin (I) or peptides (II), or digestive enzymes(III) to an individual having the disorder, to improve a symptom of the disorder.

ACTIVITY - Antiparkinsonian; Hypotensive; Antitumor; Antiarrhythmic; Antiinflammatory; Protozoacide; Antidiabetic.

A 6 year old male child previously diagnosed with Familial Dysautonomia who presented with marked autonomic dysfunction, including a total inability to walk or talk and lacked fine motor movements, and underwent an autonomic crisis 5-7 times per day, which necessitated continuous skilled nursing, with life support equipment including a respirator in close proximity, was administered ongoing secretin infusions. A test dose of 1 U of, e.g., Secretin-Ferring was administered to the individual. approximately one minute after infusion, the individual was examined for signs of allergic reaction including rash, increased heart rate, and increase of blood pressure. If the individual did not display any signs of allergic reaction, the remaining units of Secretin-Ferring was administered to the individual. The individual

received a 1-2 U/kg of body weight infusion of Secretin-Ferring via an IV push method approximately every 4 weeks for 8 months. After the 4th secretin administration, the child began to exhibit significant changes in his behavior as well as significant changes in the autonomic dysfunction. The child began to walk and utter words. His loss of blood pressure and autonomic crisis became non-existent, his need for a nurse practitioner was completely eliminated, and he was able to work with an aide who helped him ambulate.

MECHANISM OF ACTION - None given.

USE - M1 is useful for determining if an individual has, or can develop, a DD or condition, where the quantity of the compound in the **stool** as determined by the analyzing step is indicative of abnormal pancreatic function or abnormal protein digestion and metabolism, or inflammatory process.

M2 is useful for treating a **dysautonomic disorder** (DD) which comprises Familial dysautonomia (Riley-Day Syndrome), Parkinson's disease, catecholamine dysfunction, Hydroxylase deficiency, familial paraganglioma syndrome, aromatic-L-amino acid decarboxylase deficiency, Menke's disease, tetrahydrobiopterin deficiency, monoamine oxidase deficiency state, catecholamine type tumor or lesion as a pheochromocytoma chemodectina or neuroblastoma, Hereditary Sensory and autonomic neuropathy type III (HSAN III), central autonomic disorder type, multiple system atrophy (Shy-Drager syndrome), orthostatic intolerance syndrome, mitral valve prolapse, postural tachycardia syndrome (POTS), idiopathic hypovolemia, dopamine metabolism, cardiovascular system, hypertension, Guillain-Barre syndrome (acute idiopathic polyneuropathy), Chaga's disease, pure autonomic failure, diabetic autonomic failure, mitochondrial disease, syncope, renal disease, fetal fatal insomnia, Sudden Infant Death Syndrome (SIDS) (all claimed).

DESCRIPTION OF DRAWING(S) - The figure shows the diagram of a family tree illustrating correlation between dysautonomic conditions and other disorders.

Dwg.1/3

L8 ANSWER 5 OF 31 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2002-690118 [74] WPIDS
DOC. NO. NON-CPI: N2002-544343
DOC. NO. CPI: C2002-195013
TITLE: Determining a disorder or condition e.g., Parkinson's disease, comprises analyzing a **stool** sample to determine presence of a pathogen e.g., Giardia and Cryptosporidium and correlating it with a disorder or lack of disorder.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): FALLON, J M
PATENT ASSIGNEE(S): (FALL-I) FALLON J M
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002081628	A1	20020627	(200274)*		9

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002081628	A1 Provisional	US 2000-249239P	20001116
		US 2001-990909	20011116

PRIORITY APPLN. INFO: US 2000-249239P 20001116; US 2001-990909 20011116
AN 2002-690118 [74] WPIDS
AB US2002081628 A UPAB: 20021118
NOVELTY - Determining (M) if an individual has, or can develop, a disorder or condition, comprises obtaining a **stool** sample from the individual, analyzing the **stool** sample to determine the presence of a pathogen, and correlating the presence of a pathogen with a disorder or lack of disorder.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a **biological marker** (I) for determining if an individual has, or can develop, a disorder or condition, comprising a pathogen in a **stool** sample of the individual.

USE - (M) is useful for determining if an individual has, or can develop, a disorder or condition, where the disorder comprises a **pervasive development disorder** (**PDD**), a **dysautonomic disorder**, or a neurological disorder (claimed). (M) is useful for aiding in the diagnosis of various human disorders, such as **PDD**, dysautonomia, Parkinson's syndrome, sudden infant death syndrome (SIDS), etc.

ADVANTAGE - No data existed previously to show a correlation and association between various disorders such as e.g., **autism**, Parkinson's, ADD (attention deficit disorder), dysautonomia, and the presence of pathogens in an individuals digestive tract.
Dwg.0/4

L8 ANSWER 6 OF 31 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-215830 [27] WPIDS
CROSS REFERENCE: 2000-237538 [20]; 2002-163306 [21]; 2002-205549 [26]
DOC. NO. CPI: C2002-065947
TITLE: Method for treating or preventing a sexual dysfunction e.g. erectile dysfunction, cerebral function disorder e.g. dementia, Parkinson's disease or restless leg syndrome comprises administering a sibutramine metabolite.
DERWENT CLASS: B03 B05
INVENTOR(S): FANG, Q K; JERUSSI, T P; SENANAYAKE, C H
PATENT ASSIGNEE(S): (FANG-I) FANG Q K; (JERU-I) JERUSSI T P; (SENA-I) SENANAYAKE C H; (SEPR-N) SEPRACOR INC
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002010198	A1	20020124	(200227)*		24
WO 2002060424	A2	20020808	(200262)	EN	

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW

US 6476078 B2 20021105 (200276)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002010198	A1 CIP of	US 1999-372158	19990811

		CIP of	US 2000-662135	20000914
			US 2001-770663	20010129
WO 2002060424	A2		WO 2002-US2040	20020123
US 6476078	B2	CIP of	US 1999-372158	19990811
		CIP of	US 2000-662135	20000914
			US 2001-770663	20010129

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 6476078	B2	CIP of	US 6331571
		CIP of	US 6339106

PRIORITY APPLN. INFO: US 2001-770663 20010129; US 1999-372158 19990811; US 2000-662135 20000914

AN 2002-215830 [27] WPIDS

CR 2000-237538 [20]; 2002-163306 [21]; 2002-205549 [26]

AB US2002010198 A UPAB: 20021125

NOVELTY - A method (I) of treating or preventing sexual dysfunction, cerebral function disorder or restless leg syndrome which comprises administering to a patient a sibutramine metabolite or its salt, solvate, hydrate, clathrate or prodrug.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) A pharmaceutical composition (II) comprising:

(a) a sibutramine metabolite or its salt, solvate, hydrate, clathrate or prodrug; and

(b) a phosphodiesterase inhibitor.

(2) A lactose free pharmaceutical composition (III) comprising

(1) a sibutramine metabolite;

(2) a phosphodiesterase inhibitor; and

(3) an excipient.

ACTIVITY - Vasotropic; Nootropic; Neuroprotective; Nephrotropic; Antiparkinsonian; Anticonvulsant; Neuroleptic; Anorectic; Osteopathic; Antiarthritic; Antiinflammatory; Antismoking; Antimigraine; Tranquilizer; Analgesic; Antidiabetic; Hypertensive; Cytostatic; Cardiant; Antigout; Anitlipemic; Antithyroid; Uropathic; Antialcoholic.

MECHANISM OF ACTION - Neuronal monoamine (e.g. dopamine, serotonin (5-HT) and norepinephrine) uptake or reuptake inhibitor.

The binding affinity of (R)-desmethylsibutramine ((R)-desMe) (A) was determined at the human recombinant norepinephrine (NE) uptake site and the beta -receptor from rat adipose tissue. (A) was tested initially at 10 mu m in duplicate and was further tested at 10 different concentrations. IC50 values were determined. (A) showed IC50 of 4 nM.

USE - For treating or preventing a sexual dysfunction in male or female (preferably erectile dysfunction in male); a cerebral function disorder e.g. senile dementia, Alzheimer's type dementia, memory loss, amnesia/amnestic syndrome, disturbance of consciousness, coma, lowering of attention, speech disorders, Parkinson's disease, Lennox syndrome, **autism**, epilepsy, hyperkinetic syndrome, or schizophrenia; and for treating or preventing restless leg syndrome (claimed).

Also for treating disorders from eating disorders such as weight gain and obesity (e.g. cancer, hypertension (e.g. pulmonary hypertension), cardiovascular disease (dyslipidemia and carotid intimal medial thickening), hiatial hernia, gout, thyroid disease (e.g. diabetes), gastro-esophogeal reflux disease, infertility); platelet adhesion; apnea; obsessive-compulsive disorders, affective disorder (e.g. attention deficit disorder and attention deficit hyperactivity disorder, bipolar and manic conditions, dysthymic disorder, cyclothymic disorder), depression,

anxiety, osteoarthritis, irritable bowel syndrome, substance abuse including nicotine addiction from cigarette smoking or chewing tobacco and cocaine, alcohol addiction; migraine, chronic pain, pain such as neuropathic pain, diabetic neuropathy, chronic disorders such as premenstrual syndrome, incontinence, cerebral function disorders; chronic disorders (e.g. narcolepsy, chronic fatigue syndrome, seasonal affective disorder, fibromyalgia, premenstrual syndrome) and incontinence, insomnia, anorexia, decreased energy, and lipido and abnormal hormonal circadian rhythms.

For treating infectious diarrhea, oily fecal spotting, flatus with discharge, fecal urgency, fatty/oily **stool**, oily evacuation, increased defecation, anal leakage, and fecal incontinence. The other sexual disorders are vaginal dryness, psychosexual dysfunction, lack of sexual excitement or inability to obtain organism. The other cerebral function disorders are cerebrovascular diseases e.g. cerebral infarction, cerebral bleeding, cerebral arteriosclerosis, cerebral venous thrombosis and head injuries.

ADVANTAGE - The composition inhibits neuronal monoamine (e.g. dopamine, serotonin and norepinephrine) uptake or reuptake and provides fewer of the adverse effects associated with sibutramine.
Dwg.0/0

L8 ANSWER 7 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:537158 BIOSIS
DOCUMENT NUMBER: PREV200200537158
TITLE: Dementia in Parkinson disease: A proton magnetic resonance spectroscopy study.
AUTHOR(S): Summerfield, Christopher; Gomez-Anson, Beatriz; Tolosa, Eduardo (1); Mercader, Jose M.; Marti, M. Jose; Pastor, Pau; Junque, Carme
CORPORATE SOURCE: (1) Department of Neurology, ICMSN, Hospital Clinic, Casanova 143, 08036, Barcelona: etolosa@clinic.ub.es Spain
SOURCE: Archives of Neurology, (September, 2002) Vol. 59, No. 9, pp. 1415-1420. http://www.archneurol.com. print. ISSN: 0003-9942.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Background: Magnetic resonance spectroscopy has been shown to be useful in differentiating idiopathic Parkinson disease (PD) from atypical parkinsonian syndromes such as progressive supranuclear palsy, multiple system atrophy, and corticobasal degeneration. Objective: To systematically investigate the utility of proton magnetic resonance spectroscopy in distinguishing between idiopathic PD with dementia (**PDD**) and without dementia. Design: Group comparisons and correlations of brain metabolites with clinical and neuropsychological variables. Patients and Methods: Metabolite concentrations were acquired from voxels localized to the basal ganglia and occipital cortex in 14 patients diagnosed as having idiopathic **PDD**, 12 patients with PD without dementia, and 13 matched control subjects. The 3 groups underwent clinical and neuropsychological assessment. Results: In the occipital region, N-acetylaspartate levels were significantly reduced in the **PDD** group relative to the PD and control groups. N-acetylaspartate values correlated with neuropsychological performance but not with severity of motor impairment. Conclusions: N-acetylaspartate reduction in occipital lobes may be a marker for dementia in PD. The distribution of metabolite reduction differs from that reported in Alzheimer disease. These findings suggest that proton spectroscopy may serve as a metabolic marker of cognitive disturbance in patients with PD.

L8 ANSWER 8 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2002:625119 BIOSIS
DOCUMENT NUMBER: PREV200200625119
TITLE: SELDI-TOF mass spectrometry as a tool for **biomarker** discovery in **autism**.
AUTHOR(S): Walker, S. J. (1); Xu, A. S. L.; Vrana, K. E. (1)
CORPORATE SOURCE: (1) Dept Physiology/Pharmacology, Wake Forest Univ Sch Medicine, Winston-Salem, NC USA
SOURCE: American Journal of Human Genetics, (October, 2002) Vol. 71, No. 4 Supplement, pp. 504. http://www.journals.uchicago.edu/AJHG/home.html. print. Meeting Info.: 52nd Annual Meeting of the American Society of Human Genetics Baltimore, MD, USA October 15-19, 2002 American Society of Human Genetics . ISSN: 0002-9297.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 9 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002449780 EMBASE
TITLE: [Communication disorders: Differential diagnosis]. TRASTORNOS DE LA COMUNICACION: DIAGNOSTICO DIFERENCIAL.
AUTHOR: Campos-Castello J.; Briceno-Cuadros S.
CORPORATE SOURCE: Dr. J. Campos-Castello, Servicio de Neuropediatria, Hosp. Clin. Universitario San Carlos, Martin Lagos, s/n, E-28040 Madrid, Spain. jcampos@hcsc.insalud.es
SOURCE: Revista de Neurologia, (1 Jul 2002) 35/1 (36-44). Refs: 48
ISSN: 0210-0010 CODEN: RVNRAA
COUNTRY: Spain
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 008 Neurology and Neurosurgery
032 Psychiatry
LANGUAGE: Spanish
SUMMARY LANGUAGE: English; Spanish; Portuguese
AB Objective. To evaluate components of clinical semiology in the differential diagnosis of communication disorders (TC) and their possible biological markers. We consider two groups, according to the communication disorders themselves and their effects on social interaction. In the first case both aspects are affected in parallel and in the second it is predominantly social interaction which is affected. Development. In the first groups we studied dyslalias, dyrhrythmias, acquired aphasias, TC relation to epilepsy, types of seizures and EEG discharges. The dysphasia of development and epilepsy may be associated by chance, as a result of the same cause or the epilepsy be responsible for the TC, either because of seizures or continuously (acquired epileptic-aphasia, SLK). Based on personal data and the literature we studied the semiology, possible biological markers and differential diagnosis. We consider disorders of neurone migration and metabolic alterations of initial neuropsychological semiology and cerebellar anomalies involved in cognitive functions. In the second group we assessed **autism**, generalized disorders of development and particular syndromes with semantic-pragmatic TC. Conclusions. The development of language cannot be separated from other aspects of neurological maturation. One cannot affirm that there is a direct relationship between epilepsy and TC, although this does occur in some cases. We accept the hypothesis that SLK, POCSL and atypical EPB are clinical forms of the same syndrome of epilepsy. Recognition of the cognitive-affective cerebellar syndrome by its involvement in social executive function, language and personality characterizes certain conditions (Williams, Asperger, fragile-X, **autism**). A progressive rational battery of complementary studies on clinical data is

essential to determine biological markers in syndromes which still lack them.

L8 ANSWER 10 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002046846 EMBASE
TITLE: New centers to focus on austism and other developmental disorders.
AUTHOR: Wakefield J.
SOURCE: Environmental Health Perspectives, (2002) 110/1 (A20-A21). ISSN: 0091-6765 CODEN: EVHPAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
017 Public Health, Social Medicine and Epidemiology
021 Developmental Biology and Teratology
032 Psychiatry
046 Environmental Health and Pollution Control
052 Toxicology
LANGUAGE: English

L8 ANSWER 11 OF 31 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002437893 MEDLINE
DOCUMENT NUMBER: 22161938 PubMed ID: 12173102
TITLE: Gastrointestinal microflora studies in late-onset **autism**.
AUTHOR: Finegold Sydney M; Molitoris Denise; Song Yuli; Liu Chengxu; Vaisanen Marja-Liisa; Bolte Ellen; McTeague Maureen; Sandler Richard; Wexler Hannah; Marlowe Elizabeth M; Collins Matthew D; Lawson Paul A; Summanen Paula; Baysallar Mehmet; Tomzynski Thomas J; Read Erik; Johnson Eric; Rolfe Rial; Nasir Palwasha; Shah Haroun; Haake David A; Manning Patricia; Kaul Ajay
CORPORATE SOURCE: Infectious Diseases Section, Veterans Affairs Medical Center, West Los Angeles, CA, USA.. sidfinegol@aol.com
SOURCE: CLINICAL INFECTIOUS DISEASES, (2002 Sep 1) 35 (Suppl 1) S6-S16.
Journal code: 9203213. ISSN: 1537-6591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020829
Last Updated on STN: 20020830
Entered Medline: 20020829

AB Some cases of late-onset (regressive) **autism** may involve abnormal flora because oral vancomycin, which is poorly absorbed, may lead to significant improvement in these children. Fecal flora of children with regressive **autism** was compared with that of control children, and clostridial counts were higher. The number of clostridial species found in the **stools** of children with **autism** was greater than in the **stools** of control children. Children with **autism** had 9 species of Clostridium not found in controls, whereas controls yielded only 3 species not found in children with **autism**. In all, there were 25 different clostridial species found. In gastric and duodenal specimens, the most striking finding was total absence of non-spore-forming anaerobes and microaerophilic bacteria from control children and significant numbers of such bacteria from children with **autism**. These studies demonstrate significant alterations in the upper and lower intestinal flora of children with

late-onset **autism** and may provide insights into the nature of this disorder.

L8 ANSWER 12 OF 31 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-066037 [09] WPIDS
DOC. NO. CPI: C2002-019592
TITLE: Existence of an alternate glucose pathway for treating mental and neurological disorders is proved by considering cerebral spinal fluid as the major component.
DERWENT CLASS: B03 B04 K08
INVENTOR(S): MORITA, K
PATENT ASSIGNEE(S): (MORI-I) MORITA K
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2001046470	A1	20011129	(200209)*		9

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2001046470	A1 Provisional	US 2000-202967P	20000510
		US 2001-854298	20010509

PRIORITY APPLN. INFO: US 2000-202967P 20000510; US 2001-854298 20010509
AN 2002-066037 [09] WPIDS
AB US2001046470 A UPAB: 20020208
NOVELTY - Existence of an alternate glucose pathway is proved by:
(a) testing a small sample of cerebral spinal fluid (CSF), blood and urine for concentrations of glucose and a CSF component;
(b) infusing the CSF component into a vein;
(c) repeating step (a); and
(d) comparing the concentration of glucose and the CSF component before and after infusion into the vein.
DETAILED DESCRIPTION - Existence of an alternate glucose pathway is proved by:
(a) testing a small sample of cerebral spinal fluid (CSF), blood and urine for concentrations of glucose and a CSF component having as properties an equal ability to flow within the blood brain barrier (BBB);
(b) infusing the CSF component into a vein;
(c) repeating step (a) after infusion; and
(d) comparing the concentration of glucose and the CSF component before and after infusion into the vein.
INDEPENDENT CLAIMS are included for the following:
(i) proving the existence of an alternate glucose pathway in non-human primates involving introducing an Ommaya reservoir into the intraventricular spaces of the brain of an animal, starting a positron emission tomography (PET) scan on the animal, drawing an amount of CSF from the reservoir and equal amount of a radiolabeled compound to be injected into the reservoir and injecting the radioactive compound into the reservoir until the PET scan detects radioactivity in the surface of the cortex in the brain and the spine;
(ii) establishing a diagnostic profile for each disorder at different age groups based on observing the effect of an artificially induced chemical balance in CSF, brain, spine and plasma involving:
(1) introducing an Ommaya reservoir into an anterior horn of a

lateral ventricle of the brain, a subarachnoid catheter in the brain, a spinal catheter on the spine, intracranial pressure catheter at the CSF, a Foley catheter at a bladder, a central catheter in either a jugular or femoral vein of an animal;

(2) starting a PET scan on the animal;

(3) drawing CSF from the reservoir, subarachnoid catheter of a normal functioning brain and from the spinal region of a normal functioning spine;

(4) drawing blood from either the jugular or femoral vein;

(5) drawing urine from the bladder;

(6) measuring each **biological marker**/s present in the CSF namely glucose, metabolites, neurotransmitters, neuropeptides, insulin, immune globulins, neuronal growth factors, counter regulatory hormones, thyroid hormones, other hormones found in the CSF and peptidases on the CSF, plasma and urine drawn above for a baseline data;

(7) measuring electroencephalogram activities of the brain;

(8) measuring intracranial pressure at the CSF;

(9) injecting a radiolabeled **biological marker** into the reservoir for the determination of time before a first sample is taken for testing;

(10) infusing five times the baseline amount as determined for each of the **biological marker** from step (f) into the reservoir over a 6 hour period to artificially induce a diseased state;

(11) continuing the infusion at this elevated amount for each 6 hour interval for a total of one week;

(12) withdrawing a CSF sample from the reservoir and the subarachnoid and spinal catheters equivalent in amount as the infused volume in step (9) for the first sample and step (10) for the subsequent samples, urine from the Foley catheter and plasma from the central catheter at the time the radiolabeled **biological marker** reaches the cortex and every six hours at the completion of infusion of each **biological marker**;

(13) determining the effect of each infusion of a **biological marker** from step (10) to the level of all the **biological marker** at those time intervals;

(14) graphing for each time interval taken, the levels of the different biological markers at each infusion of a **biological marker** from step (10) for each age group; and

(15) repeating the step (1)-(14) for each **biological marker** to obtain a diagnostic profile on the effect of each **biological marker** infused at five times the normal level on the level of all the biological markers;

(iii) using a diagnostic profile obtained from an animal (preferably non-human primates) for diagnosing a human patient involving:

(A) drawing a normal human CSF sample by a spinal tap from different age groups for determining a normal level for each biological marker at the different age groups;

(B) drawing human CSF samples by a spinal tap from patients from different age groups with a disorder for determining a diseased level for each biological marker at the different age groups;

(C) graphing the levels of the different biological markers obtained from the normal and the diseased patients;

(D) discarding the biological marker where the normal level of the animal is different from the normal level of the human patient;

(E) comparing the graph of the normal patient and the graph of the diseased patient with the graph obtained from the animal;

(F) choosing the graph of the animal that comes closest to the graph of the normal and the diseased patient;

(G) determining the biological marker in the animal that produced the same graph as the diseased human patient; and

(H) diagnosing a disorder of the diseased human patient based on the biological marker determined from step (G); and

(iv) a medication having a property of crossing BBB and any one of the properties selected from binding a biological marker present in excess in CSF to reduce the level to normal or to eliminate the level to undetectable amounts or elevating a level of the biological marker to normal and introducing an absent biological marker in CSF.

ACTIVITY - Nootropic; Antidiabetic; Neuroprotective.

MECHANISM OF ACTION - None given.

USE - For testing, diagnosing and treating mental and neurological disorders not detectable by Computed Tomography (CT) scan and Magnetic Resonance Imaging (MRI) (claimed).

ADVANTAGE - The alternate glucose pathway when applied in conjunction with other pathways explains the motor function, the function of memory, mind and emotion, the effect of the thyroid hormones to mental disorders, dementia in cerebral ischemic infarction, hypoglycemia, Alzheimer disease, autism, dementia and psychiatric illness. Also provides effective medications or facilitate the isolation or synthesis of effective medications.

Dwg.0/2

L8 ANSWER 13 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001229617 EMBASE
TITLE: Recent discoveries affecting drug therapy.
SOURCE: Drug Benefit Trends, (2001) 13/6 (62+65-66).
ISSN: 1080-5826 CODEN: DBTRFN
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
004 Microbiology
030 Pharmacology
032 Psychiatry
029 Clinical Biochemistry
046 Environmental Health and Pollution Control
LANGUAGE: English

L8 ANSWER 14 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2002:178868 BIOSIS
DOCUMENT NUMBER: PREV200200178868
TITLE: Pediatric autoimmune neuropsychiatric disorders associated with streptococci: Rheumatic fever/sydenham chorea, Tourette Syndrome, and **autism**.
AUTHOR(S): Moore, J. C. (1); Augustine, N. H. (1); Hill, H. R. (1); McMahon, W. M. (1)
CORPORATE SOURCE: (1) Departments of Psychiatry, Pediatrics, Pathology, and Medicine, University of Utah School of Medicine, Salt Lake City, UT USA
SOURCE: Journal of Investigative Medicine, (January, 2001) Vol. 49, No. 1, pp. 49A. http://www.jinvmed.com/. print.
Meeting Info.: Joint Regional Meeting of the Western Section American Federation for Medical Research, the Western Society for Clinical Investigation and the Western Association of Physicians Carmel, California, USA February 07-10, 2001
ISSN: 1081-5589.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 15 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2000:482366 BIOSIS
DOCUMENT NUMBER: PREV200000482366
TITLE: Secretin improves intestinal permeability in autistic children (AC.
AUTHOR(S): Horvath, Karoly (1); Zielke, Ronald H. (1); Collins, Roger M. (1); Rabsztyn, Anna (1); Medeiros, Lisa A. (1); Perman, Jay (1)
CORPORATE SOURCE: (1) Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD USA
SOURCE: JPGN, (2000) Vol. 31, No. Supplement 2, pp. S30-S31. print. Meeting Info.: World Congress of Pediatric Gastroenterology, Hepatology, and Nutrition Boston, Massachusetts, USA August 05-09, 2000
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L8 ANSWER 16 OF 31 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000103813 MEDLINE
DOCUMENT NUMBER: 20103813 PubMed ID: 10638458
TITLE: Research on screening and diagnosis in **autism**: a work in progress.
COMMENT: Comment in: J Autism Dev Disord. 2000 Dec;30(6):625
AUTHOR: Bristol-Power M M; Spinella G
CORPORATE SOURCE: National Institute of Child Health and Human Development, Bethesda, Maryland, USA.. BRISTOLM@mail.nih.gov
SOURCE: JOURNAL OF AUTISM AND DEVELOPMENTAL DISORDERS, (1999 Dec) 29 (6) 435-8. Ref: 11
Journal code: 7904301. ISSN: 0162-3257.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000309
Last Updated on STN: 20030105
Entered Medline: 20000218

AB In June 1998, the National Institutes of Health **Autism** Coordinating Committee (NIH/ACC) invited representatives of 13 major medical and other professional academies and associations and six national **autism** parent research organizations to review research data on screening and diagnosis of **autism** spectrum disorders. Ten review papers and more than 4,000 publications were consulted in this effort. This paper highlights some promising areas for research identified in this process. One of the highest priorities is the search for the ultimate diagnostic indicator, a **biological marker**(s), for example, genetic, metabolic, immunologic, neurologic, that will distinguish **autism** unequivocally from other developmental disabilities. In the interim, research on infant screening and diagnosis might lower the threshold age for diagnosis to 8-12 months. The role of sensory-motor disorders in early diagnosis needs further research. Earlier and better diagnosis of co-occurring, potentially treatable disorders, including epileptic and epileptiform disorders, has implications both for diagnosis and treatment. Pharmacogenetic and pharmacogenomic research strategies could help diagnose subtypes and responders versus nonresponders to potential treatments. Better endpoints and outcome measures are needed, including improved procedures for cognitive and neuropsychological testing, more

knowledge about verbal and nonverbal communication milestones, and less invasive and more sensitive neuroimaging measures. Critical questions remain regarding regression after apparently normal development, and about possible environmental precipitants. Finally, field trials of the reliability and validity of screening and diagnosis using the newly developed practice guidelines are needed.

L8 ANSWER 17 OF 31 MEDLINE
ACCESSION NUMBER: 1998182962 MEDLINE
DOCUMENT NUMBER: 98182962 PubMed ID: 9522491
TITLE: [Platelet serotonin as a **biological marker** of **autism**].
Serotonina plaquetaria como marcador biologico de **autismo**.
AUTHOR: Levy P Q; Bicho M P.
CORPORATE SOURCE: Laboratorio de Genetica, Faculdade de Medicina de Lisboa, Universidade de Lisboa.
SOURCE: ACTA MEDICA PORTUGUESA, (1997 Dec) 10 (12) 927-31.
Journal code: 7906803. ISSN: 0870-399X.
PUB. COUNTRY: Portugal
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Portuguese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19990129
Entered Medline: 19980513

AB Platelet levels of serotonin were determined by a quantitative direct radioimmunoassay, in a group of autistic patients and a control group. Thirty six autistic patients (28 males and 8 females), all with severe mental retardation, and a group of 23 matched controls, were studied. The serotonin levels in autistic patients (mean +/- SD) (88.37 mmol/dl +/- 40.38) were significantly higher that in the control group (49.54 mmol/dl +/- 16.49). There were no significant differences in levels between the sexes and age groups among subjects in the patient and the control groups. We detected a hyperserotoninaemia in 70% of the autistic patients. We also discuss correlations between serotonin levels in our patients with known aetiologies and levels quoted in the literature and propose RIA to be used as a quick, easy and reliable method for the analysis of large numbers of samples.

L8 ANSWER 18 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 95232540 EMBASE
DOCUMENT NUMBER: 1995232540
TITLE: Is decreased blood plasma concentration of the complement C4B protein associated with attention-deficit hyperactivity disorder?.
AUTHOR: Warren R.P.; Odell J.D.; Warren W.L.; Burger R.A.; Maciulis A.; Torres A.R.
CORPORATE SOURCE: UMC 6895, Utah State University,Logan, UT 84322, United States
SOURCE: Journal of the American Academy of Child and Adolescent Psychiatry, (1995) 34/8 (1009-1014).
ISSN: 0890-8567 CODEN: JAAPEE
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
032 Psychiatry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Objective: The complement system is a group of blood proteins that play an important role in defending against viral and bacterial infections. The objective of this investigation was to study the plasma levels of the C4B protein in attention-deficit hyperactivity disorder (ADHD) in an attempt to associate infections with the development of some cases of this disorder. Method: C4B plasma protein levels were studied using an enzyme-linked immunosorbent assay in a group of 23 subjects meeting DSM-III-R criteria for ADHD and a similar number of age- and sex-matched controls. Also studied were parents of the ADHD subjects. Results: C4B plasma levels (157.0 .mu.g/mL) in the ADHD subjects were significantly (p < .01) lower than those (239.3 .mu.g/mL) in the normal age-matched subjects. Mothers of the ADHD subjects also had significantly lower C4B values compared with mothers of normal children. On the other hand, C4B values in the fathers were not significantly altered. Conclusions: Decreased C4B levels in ADHD, if replicated, may represent an important marker for ADHD (or a subgroup of ADHD). It also seems plausible that C4B levels are an important etiological factor for ADHD.

L8 ANSWER 19 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93134638 EMBASE
DOCUMENT NUMBER: 1993134638
TITLE: Serotonin and amino acid content in platelets of autistic children.
AUTHOR: Rolf L.H.; Haarmann F.Y.; Grotemeyer K.-H.; Kehrer H.
CORPORATE SOURCE: Department of Neurology, Westfalische Wilhelms-Universitat, Albert-Schweitzer-Strasse 33,D-4400 Munster, Germany
SOURCE: Acta Psychiatrica Scandinavica, (1993) 87/5 (312-316). ISSN: 0001-690X CODEN: APYSA
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 032 Psychiatry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The platelet levels of serotonin and the amino acids aspartic acid, glutamine, glutamic acid and gamma-aminobutyric acid were measured in 18 drug-free autistic (DSM-III criteria) and 14 age-matched healthy children. Serotonin was significantly increased while the amino acids aspartic acid, glutamine, glutamic acid and gamma-aminobutyric acid were significantly decreased in comparison with the controls. It is suggested that the decline of the amino acids in platelets from autistic children represents a biochemical marker related to infantile **autism**.

L8 ANSWER 20 OF 31 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 94050412 MEDLINE
DOCUMENT NUMBER: 94050412 PubMed ID: 8232778
TITLE: Exclusion of linkage of genetic focal sharp waves to the HLA region on chromosome 6p in families with benign partial epilepsy with centrotemporal sharp waves.
AUTHOR: Whitehouse W; Diebold U; Rees M; Parker K; Doose H; Gardiner R M
CORPORATE SOURCE: Department of Paediatrics, University College London Medical School, UK.
SOURCE: NEUROPEDIATRICS, (1993 Aug) 24 (4) 208-10. Journal code: 8101187. ISSN: 0174-304X.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940117

Last Updated on STN: 20020125
Entered Medline: 19931203

AB Benign partial epilepsy with centrotemporal sharp waves (benign rolandic epilepsy, BRE) is a common form of idiopathic, localisation-related epilepsy of childhood. The characteristic age-dependent focal sharp wave (fsw) found on the EEG in this disorder segregates as an autosomal dominant trait in families with probands with BRE and acts as a **neurobiological marker** for the increased risk of developing BRE, other benign partial epilepsies of **childhood**, and other **developmental disorders** in these families. One of the genes for idiopathic generalised epilepsy (IGE), designated EJM1, has been mapped in families with probands with juvenile myoclonic epilepsy, by linkage to the HLA region on chromosome 6. As BRE and IGE are benign, idiopathic, age-dependent epilepsies, EJM1 is a candidate locus for the fsw underlying BRE and related disorders. Genetic linkage analysis was undertaken in 11 families with probands with BRE and one or more first degree relatives with fsw, with or without BRE, using a polymorphic DNA marker within the HLA region. Apparently unaffected individuals were classed as affection status unknown. Assuming autosomal dominant inheritance with a penetrance of 0.9 gave a lod score of -2.3 at zero recombination, excluding the candidate gene region around HLA. These observations exclude an important candidate gene for this common disorder, and suggest a fundamental molecular and genetic distinction between the benign partial epilepsies of childhood and the idiopathic generalised epilepsies.

L8 ANSWER 21 OF 31 JICST-EPlus COPYRIGHT 2003 JST
ACCESSION NUMBER: 920679514 JICST-EPlus
TITLE: Study of the background host factors in methicillin-resistant Staphylococcus aureus(MRSA)-induced infectious disease after surgery of the digestive cancer, particularly in MRSA-induced enteritis.
AUTHOR: MIYAKE HIROSHI; KUROSU YASUHIKO; TAKIZAWA HIDEHIRO; UGAJIN WAKATO; SHIBATA MASAHIKO; AMANO SADAO
CORPORATE SOURCE: Nihon Univ., School of Medicine
SOURCE: Biotherapy (Tokyo), (1992) vol. 6, no. 9, pp. 1360-1364.
Journal Code: L0028A (Fig. 3, Tbl. 2, Ref. 8)
ISSN: 0914-2223
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
LANGUAGE: Japanese
STATUS: New

AB In recent years, postoperative methicillin-resistant Staphylococcus aureus(MRSA)-induced enteritis due to the increase of MRSA-induced infectious disease has been clinically problematical in the digestive surgey field. It is known that this disease occurs frequently after surgery of the upper digestive tract in the aged with decreased nutrition and immunity. In order to clarify the background host factors of the onset of MRSA-induced enteritis after surgery for digestive cancer and to obtain aid for the prevention of the disease, the preoperative conditions of nutrition and immunity in patients with digestive cancer were retrospectively studied, while dividing them into onset and non-onset groups of enteritis. Sixtyone cases with digestive cancer underwent surgery in the First Department of Surgery during the past 1 year, and MRSA-induced enteritis developed post-operatively in 11 cases. No relationships were found between this disease and age, preoperative throat, and **stool** culture, the presence or absence of blood transfusion or gastric acidity. However, serum levels of transferrin, retinol binding protein and IAP were significantly differentbetween the two, and the disease was seen more frequently in patients with negative

PDD skin test. From these facts, it was considered that improvement of the preoperative conditions such as nutrition and immune functions using intravenous hyperalimentation helps to prevent the onset of this disease. (author abst.)

L8 ANSWER 22 OF 31 MEDLINE
ACCESSION NUMBER: 92022254 MEDLINE
DOCUMENT NUMBER: 92022254 PubMed ID: 1925380
TITLE: [Rett syndrome. A well defined but mysterious encephalopathy].
Le syndrome de Rett. Une encephalopathie bien individualisee mais mysterieuse.
AUTHOR: Arzimanoglou A
CORPORATE SOURCE: Service de pediatrie, hopital de la Salpetriere, Paris.
SOURCE: REVUE DU PRATICIEN, (1991 Sep 15) 41 (20) 1940-4. Ref: 17
Journal code: 0404334. ISSN: 0035-2640.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Foreign
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911108

AB The Rett syndrome is characterized by a progressive development of loss of intellectual functions and of motricity, including abnormal stereotypic hand movements and reduction of the motor skill. This syndrome is exclusively observed in girls. Its typical evolution is characterized by a normal initial development (until 6 to 18 months after birth) followed by a progressive installation of the clinical signs in 4 steps. There is currently no **biological marker** for the Rett syndrome and therefore the diagnosis is only based on clinical criteria. The most common erroneous diagnosis is infantile **autism**. In this review, the current status of clinical, genetic and pathogenetic knowledge of the Rett syndrome is presented.

L8 ANSWER 23 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1992:237595 BIOSIS
DOCUMENT NUMBER: BA93:125620
TITLE: DIFFERENT TYPES OF ONSET IN RETT'S SYNDROME.
AUTHOR(S): GASSIO R; PINEDA M; CAMPISTOL J; COLOMER J; CONILL J; POO P; VERNET A; SANMARTI F X; FERNANDEZ-ALVAREZ E
CORPORATE SOURCE: SERV. DE NEUROPEDIATRIA, HOSP. SAN JUAN DE DIOS, CTRA. DE ESPLUGAS, S/N, 08034 BARCELONA.
SOURCE: REV ESP PEDIATR, (1991) 47 (282), 509-514.
CODEN: REPEAW. ISSN: 0034-947X.
FILE SEGMENT: BA; OLD
LANGUAGE: Spanish

AB In 1988 The Rett Syndrome Diagnostic Criteria Work Group defined the actual diagnostic criteria for the Rett Syndrome. Besides the typical observations atypical variants have been described, being the most important "formes frustes" or incompletes and forms initiated with infantile spasms. We present 6 observations of atypical variants of the Rett Syndrome, because 1 or 2 of the necessary criteria are not followed. They were diagnosed during a period of 6 years (May-84 to May-90). In 4 out of 6 observations there aren't deceleration of head growth, through the clinical course and signs are typical of the Rett Syndrome. In conclusion clinicians should be more rigorous with the diagnostic criteria

until a **biological marker** will be found.

L8 ANSWER 24 OF 31 MEDLINE
ACCESSION NUMBER: 92061921 MEDLINE
DOCUMENT NUMBER: 92061921 PubMed ID: 1953623
TITLE: Diagnostic and assessment issues related to pharmacotherapy for children and adolescents with **autism**.
AUTHOR: Campbell M; Kafantaris V; Malone R P; Kowalik S C; Locascio J J
CORPORATE SOURCE: Department of Psychiatry, New York University Medical Center, NY 10016.
CONTRACT NUMBER: 1 T32 MH-18915 (NIMH)
MH-32212 (NIMH)
MH-40177 (NIMH)
SOURCE: BEHAVIOR MODIFICATION, (1991 Jul) 15 (3) 326-54. Ref: 150
Journal code: 7803043. ISSN: 0145-4455.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199112
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19990129
Entered Medline: 19911204

AB **Autism** involves not only developmental delays but also aberrant behavior, both of which change in nature over time. Rating instruments may be useful to assess maladaptive and adaptive behaviors of autistic children in a standardized way and, perhaps, to measure change due to treatment. With the expansion of basic science, knowledge, and technology, there is increasing evidence that **autism** is etiologically heterogeneous. Currently, there is no **biological marker** specific to **autism**, although hyperserotonemia is a consistent finding in one third of autistic children. An aim of basic science research has been to develop a rational pharmacotherapy based upon the underlying neurochemistry. However, at the present time, this approach has not always been successful. It is expected that the development and use of more restrictive criteria, delineation of subtypes of **autism**, and interaction of descriptive, behavioral, clinical, and basic research will lead to more effective planning for treatment. The relationship of assessment to treatment response is presented and discussed.

L8 ANSWER 25 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1990:336781 BIOSIS
DOCUMENT NUMBER: BA90:44800
TITLE: BRIEF REPORT COGNITIVE SUBCLINICAL MARKERS IN **AUTISM**.
AUTHOR(S): SMALLEY S L; ASARNOW R F
CORPORATE SOURCE: DEP. PSYCHIATRY, 48-421 NPI, UNIV. CALIF., LOS ANGELES, SCH. MED., 760 WESTWOOD PLAZA, LOS ANGELES, CALIF. 90024, USA.
SOURCE: J AUTISM DEV DISORD, (1990) 20 (2), 271-278.
CODEN: JADDDQ.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB **Autism** is a behaviorally defined disorder with a significantly increased chance of recurrence in families (2-3%) compared with the population frequency (0.02-.005%), suggesting genetic influences. However,

although the sibling recurrence risk is some hundredfold greater than the population risk, it is far smaller than expected if **autism** was inherited as a single gene disorder (Smalley, Asarnow, & Spence, 1988). Family and twin studies have suggested that **autism** is etiologically heterogeneous. Despite this etiological heterogeneity, many studies of cognitive abilities in **autism** have reported a relatively consistent and homogeneous profile. Specifically, autistic individuals share a cognitive profile marked by language deficits, particularly in verbal comprehension and abstraction, but intact perceptual-motor and rote memory abilities (Asarnow, Tanguay, Bott, & Freeman, 1987; Lincoln, Corchesne, & Kilman, 1988). Many family studies of **autism** are now focusing on detecting what might be inherited in **autism**, that is the identification of genetic "subclinical markers". We define a subclinical **marker** as a **biological** or behavioral measure that is presumably more proximal to the underlying gene (or genes) involved in **autism** than the clinical syndrome. To be considered a putative subclinical marker, the measure under consideration should detect differences among (a) autistic probands compared with appropriately matched normal controls, and (b) a greater proportion of relatives of autistic probands compared with normal controls.

L8 ANSWER 26 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:269905 BIOSIS
DOCUMENT NUMBER: BA86:9149
TITLE: ELECTRORETINOGRAMS IN **AUTISM** A PILOT STUDY OF B-WAVE AMPLITUDES.
AUTHOR(S): RITVO E R; CREEL D; REALMUTO G; CRANDALL A S; FREEMAN B J; BATEMAN J B; BARR R; PINGREE C; COLEMAN M; PURPLE R
CORPORATE SOURCE: NEUROPSYCHIATR. INST., 760 WESTWOOD PLAZA, LOS ANGELES, CALIF. 90024.
SOURCE: AM J PSYCHIATRY, (1988) 145 (2), 229-232. CODEN: AJPSAO. ISSN: 0002-953X.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The authors recorded electroretinograms for 27 autistic patients and 20 age- and sex-matched healthy volunteers. Thirteen (48%) of the autistic patient demonstrated subnormal b-wave amplitudes, which may indicate abnormal retinal function. One patient was tested serially at two site; his low b-wave amplitude did not vary over time or between the two sites. If this retinal finding can be confirmed at other laboratories and in larger samples of autistic patients, it might provide a marker for a specific subtype of **autism**.

L8 ANSWER 27 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 88213099 EMBASE
DOCUMENT NUMBER: 1988213099
TITLE: Biological basis of **autism**.
AUTHOR: Golden G.S.
CORPORATE SOURCE: University of Tennesse, Memphis, TN 38105, United States
SOURCE: International Pediatrics, (1988) 3/2 (110-114). ISSN: 0885-6265 CODEN: INPDEV
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
022 Human Genetics
032 Psychiatry
050 Epilepsy
LANGUAGE: English

SUMMARY LANGUAGE: English
AB **Autism**, characterized primarily by severe deficits in social and interpersonal behavior, and abnormalities in language and its use as a tool for communication, is strongly associated with clinical evidence of brain dysfunction. Especially prominent are the relationships to mental retardation and epilepsy. It is clear, however, that the autistic core is not just a manifestation of these disorders, and can exist without them. Attempts to find a specific **biological marker**, using the tools of neuroanatomy, neuropathology, neurophysiology, neurochemistry and genetics have not yet been successful. A number of comprehensive theories for the underlying pathophysiology have been developed, but none has received strong enough support to be accepted as the final answer. As newer tools for neurobiological investigations in vivo are developed, the long sought **biological marker** may yet be found.

L8 ANSWER 28 OF 31 MEDLINE
ACCESSION NUMBER: 86141347 MEDLINE
DOCUMENT NUMBER: 86141347 PubMed ID: 3456386
TITLE: Retinal pathology in autistic children--a possible **biological marker** for a subtype?.
AUTHOR: Ritvo E R; Creel D; Crandall A S; Freeman B J; Pingree C; Barr R; Realmuto G
SOURCE: JOURNAL OF THE AMERICAN ACADEMY OF CHILD PSYCHIATRY, (1986 Jan) 25 (1) 137.
Journal code: 7505568. ISSN: 0002-7138.
PUB. COUNTRY: United States
DOCUMENT TYPE: Letter
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198603
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19990129
Entered Medline: 19860328

L8 ANSWER 29 OF 31 MEDLINE
ACCESSION NUMBER: 86239358 MEDLINE
DOCUMENT NUMBER: 86239358 PubMed ID: 3087180
TITLE: Atypical forms of Rett syndrome.
AUTHOR: Goutieres F; Aicardi J
SOURCE: AMERICAN JOURNAL OF MEDICAL GENETICS. SUPPLEMENT, (1986) 1 183-94.
Journal code: 8706133. ISSN: 1040-3787.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198606
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19990129
Entered Medline: 19860627
AB In the absence of any **biological marker**, Rett syndrome (RS) is defined by clinical criteria which have been proposed at the second Vienna conference on RS and patients who do not fulfill those criteria cannot be included. However, some patients partially fulfill the criteria but lack some of the essential characteristics. Seven such patients are reported. All patients were girls. Atypical manifestations included absence of a normal development during the first months of life (5 patients), absence of deterioration (1 patient), or presence of initial and intense seizure activity (2 patients). If such cases are indeed atypical RS, the spectrum of clinical manifestations will have to be

broadened and deterioration of previously acquired skills may not be an essential requirement for its diagnosis. The exclusive occurrence of atypical and of typical cases in females suggests that both constitute a single morbid entity.

L8 ANSWER 30 OF 31 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 86048442 MEDLINE
DOCUMENT NUMBER: 86048442 PubMed ID: 3864386
TITLE: A "new" chromosome marker common to the Rett syndrome and infantile **autism**? The frequency of fragile sites at X p22 in 81 children with infantile **autism**, childhood psychosis and the Rett syndrome.
AUTHOR: Gillberg C; Wahlstrom J; Hagberg B
SOURCE: BRAIN AND DEVELOPMENT, (1985) 7 (3) 365-7.
Journal code: 7909235. ISSN: 0387-7604.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198511
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19990129
Entered Medline: 19851125

AB Chromosomes from 46 autistic, 20 psychotic and 15 Rett syndrome children were cultured in a folic-acid-depleted medium. Nine percent of the autistic, 20% of the psychotic and 40% of the Rett syndrome cases showed a "new" chromosomal anomaly, viz a fragile site at the (X) (p22) location. It is suggested that in some cases of **autism**/psychosis and the Rett syndrome, there might be a common **biological marker** for the common type of psychiatric disturbance. However, as the population frequency of the chromosome marker is not yet known, conclusions must be drawn with great caution.

L8 ANSWER 31 OF 31 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 76140283 EMBASE
DOCUMENT NUMBER: 1976140283
TITLE: Hunches on some biological factors in **autism**.
AUTHOR: Sullivan R.C.
CORPORATE SOURCE: Informat. Ref. Serv., Nat. Soc. Autist. Child., Huntington, W.Va. 25702, United States
SOURCE: Journal of Autism and Childhood Schizophrenia, (1975) 5/2 (177-184).
CODEN: JAUCB4
DOCUMENT TYPE: Journal
FILE SEGMENT: 032 Psychiatry
008 Neurology and Neurosurgery
031 Arthritis and Rheumatism
LANGUAGE: English

AB The author searches with particular intensity for clues to what may cause **autism**. She found an increasing number of families who report diagnosed rheumatoid arthritis (RA) and related conditions such as gout, high uric acid, systemic lupus erythematosus, or undiagnosed bone pain; diagnosed celiac disease and/or malabsorption gastrointestinal symptoms and such related conditions as diarrhea, colitis, abdominal distention, unusually large **stools** that float, excessive flatulence, abdominal cramps; and depression, with numerous complaints of headache and abdominal pain.

=> d ibib abs ind 1-3

L1 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:488136 HCAPLUS
DOCUMENT NUMBER: 137:30245
TITLE: Methods for diagnosing pervasive development disorders, dysautonomia and other neurological conditions
INVENTOR(S): **Fallon, Joan M.**
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 9 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002081628	A1	20020627	US 2001-990909	20011116
PRIORITY APPLN. INFO.:			US 2000-249239P P	20001116

AB Methods for aiding in the diagnosis of disorders including, but not limited to, PDDs (Pervasive Development Disorders), Dysautonomic disorders, Parkinson's disease and SIDS (Sudden Infant Death Syndrome). In one aspect, a diagnosis method comprises analyzing a stool sample of an individual for the presence of a biol. marker (or marker compd.) comprising one or more pathogens, which provides an indication of whether the individual has, or can develop, a disorder including, but not limited to, a PDD, Dysautonomia, Parkinsons disease and SIDS. Preferably, the presence of one or more pathogens is detd. using a stool immunoassay to det. the presence of antigens in a stool sample, wherein such antigens are assocd. with one or more pathogens including, but not limited to, Giardia, Cryptosporidium, E. histolytica, C. difficile, Adenovirus, Rotavirus or H. pylori.

IC ICM C12Q001-70
ICS C12Q001-68; G01N033-554; G01N033-569; C12Q001-04; G01N033-53
NCL 435007100
CC 9-10 (Biochemical Methods)
Section cross-reference(s): 10, 14
ST diagnosing pervasive development disorder dysautonomia neurol
IT Disease, animal
(Dysautonomic; methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)
IT Nervous system
(disease; methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)
IT Development, mammalian postnatal
(disorder, Pervasive; methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)
IT Adenoviridae
Biomarkers (biological responses)
Clostridium difficile
Cryptosporidium
Diagnosis
Entamoeba histolytica
Feces
Giardia
Helicobacter pylori
Human
Immunoassay

Parkinson's disease
Pathogen
Rotavirus
(methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)

IT Antigens
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)

IT Death
(sudden infant death syndrome; methods for diagnosing pervasive development disorders, dysautonomia and other neurol. conditions)

L1 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:142908 HCAPLUS
DOCUMENT NUMBER: 136:180178
TITLE: Methods for diagnosing and treating dysautonomia and other dysautonomic conditions
INVENTOR(S): **Fallon, Joan M.**
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002014537	A2	20020221	WO 2001-US25343	20010814
WO 2002014537	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001084865	A5	20020225	AU 2001-84865	20010814
US 2002037284	A1	20020328	US 2001-929592	20010814
PRIORITY APPLN. INFO.:			US 2000-224991P P	20000814
			WO 2001-US25343 W	20010814

AB Methods for aiding in the diagnosis of dysautonomic disorders and dysautonomic conditions and methods for treating individuals diagnosed as having a dysautonomic disorder or a dysautonomic condition. In one aspect, a diagnosis method comprising analyzing a stool sample of an individual for the presence of a biol. marker wherein the quantity of the biol. marker is an indication of whether the individual has, or can develop, a dysuatonic disorder or dysautonomic condition, as well as a therapeutic method for treating a dysautonomic disorder or dysautonomic condition by administration of, e.g., secretin, neuropeptides, peptides and/or digestive enzymes.

IC ICM C12Q001-00
CC 9-16 (Biochemical Methods)
Section cross-reference(s): 1, 2, 7, 14
ST diagnosing treating dysautonomia dysautonomic
IT Disease, animal

(Central autonomic disorder; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Trypanosoma cruzi
(Chagas' disease from; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Diabetic autonomic failure; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Dysautonomia; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Familial dysautonomia (Riley-Day Syndrome); methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Familial paraganglioma syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Insomnia
(Fetal fatal; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Mental disorder
(Menkes' syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Mitral valve prolapse.; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Orthostatic intolerance syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Enzymes, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Pancreatic; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Pure autonomic failure; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Disease, animal
(Shy-Drager syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Reflex
(baroreceptor, failure; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Enzymes, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(digestive; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Mitochondria
(diseases; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Catecholamines, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(dysfunction; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Blood
(hypovolemia, Idiopathic; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)
IT Biomarkers (biological responses)
Cardiovascular system
Diagnosis
Digestion, biological

Drugs
Feces
Hypertension
Inflammation
Kidney, disease
Metabolism, animal
Pancreas, disease
Parkinson's disease
Pheochromocytoma
Samples
(methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Neuropeptides
Peptides, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Nervous system
(multiple system atrophy; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Nerve, neoplasm
(neuroblastoma; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Nerve, disease
(neuropathy, Hereditary Sensory and autonomic neuropathy type III (HSAN III); methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Nerve, disease
(polyneuropathy, Acute idiopathic, Gullain-Barre syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Death
(sudden infant death syndrome; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Brain, disease
(syncope; methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT Heart, disease
(tachycardia, Postural tachycardia syndrome (POTS); methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT 51-61-6, Dopamine, biological studies 9001-66-5, Monoamine oxidase 9013-38-1, Dopamine-.beta. Hydroxylase 9042-64-2, Aromatic-L-amino acid decarboxylase 17528-72-2, Tetrahydrobiopterin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT 9004-07-3, Chymotrypsin
RL: DGN (Diagnostic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

IT 1393-25-5, Secretin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods for diagnosing and treating dysautonomia and other dysautonomic conditions)

L1 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:452874 HCAPLUS
DOCUMENT NUMBER: 135:41046
TITLE: Methods for treating pervasive development disorders with secretin or digestive enzyme

INVENTOR(S): **Fallon, Joan M.**
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001043764	A2	20010621	WO 2000-US34000	20001215
WO 2001043764	A3	20011129		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6534063	B1	20030318	US 1999-466559	19991217
US 2002090653	A1	20020711	US 2001-41073	20011231

PRIORITY APPLN. INFO.: US 1999-466559 A 19991217
US 2000-707395 A 20001107

AB Disclosed is a method of utilizing the chymotrypsin level of an individual as a measure of the success of secretin, other neuropeptides, and peptides or digestive enzyme administration to such individuals, and in particular, as a prognosticative of potential secretin, other neuropeptides, peptides, and digestive enzyme administration for persons having ADD, ADHD, Autism and other pervasive development disorder (PDD)-related disorders. In one aspect, a method for detg. the efficacy of secretin, other neuropeptides, peptides, or digestive enzymes for the treatment of an individual diagnosed with a PDD comprises obtaining a sample of feces from an individual, detg. a quant. level of chymotrypsin present in the sample, and correlating the quant. level of chymotrypsin detd. to be present in the sample with the PDD to det. the efficacy of treating the individual with secretin, other neuropeptides, peptides, or digestive enzyme administration. In another aspect, a therapeutic method for treating an individual diagnosed with a PDD comprises detg. the efficiency of secretin, other neuropeptides, peptides, and digestive enzymes administration for the treatment of the individual based on a measure of the individual's chymotrypsin level, and administering secretin, other neuropeptides, peptides, or digestive enzymes to the individual based on the detn. of the measure of the individual's chymotrypsin level.

IC ICM A61K038-00
CC 1-12 (Pharmacology)
Section cross-reference(s): 2, 7, 9, 14
ST secretin digestive enzyme treatment pervasive development disorder; chymotrypsin feces efficacy treatment neuropeptide peptide; autism chymotrypsin feces treatment secretin; attention deficit disorder chymotrypsin secretin
IT Mental disorder
(attention deficit disorder; methods for treating pervasive development disorders with secretin or digestive enzyme)
IT Mental disorder
(attention deficit hyperactivity disorder; methods for treating pervasive development disorders with secretin or digestive enzyme)
IT Mental disorder

(autism; methods for treating pervasive development disorders with secretin or digestive enzyme)

IT Feces
(chymotrypsin detn. in; methods for treating pervasive development disorders with secretin or digestive enzyme)

IT Pancreas
(digestive enzyme of; methods for treating pervasive development disorders with secretin or digestive enzyme)

IT Enzymes, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(digestive; methods for treating pervasive development disorders with secretin or digestive enzyme)

IT Development, mammalian postnatal
(disorder, pervasive development disorder; methods for treating pervasive development disorders with secretin or digestive enzyme)

IT Neuropeptides
Peptides, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods for treating pervasive development disorders with secretin or digestive enzyme)

IT 9004-07-3, Chymotrypsin
RL: ANT (Analyte); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); BSU (Biological study, unclassified); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(methods for treating pervasive development disorders with secretin or digestive enzyme)

IT 1393-25-5, Secretin 9000-92-4, Amylase 9001-42-7, Maltase 9001-62-1, Lipase 9001-92-7, Protease 9002-07-7, Trypsin 9012-54-8, Cellulase 37288-39-4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(methods for treating pervasive development disorders with secretin or digestive enzyme)